

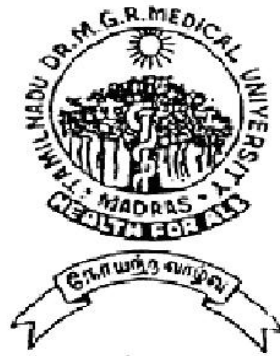
**A STUDY ON  
NON-TRAUMATIC ULCERS OF LOWER LIMB**

**A STUDY OF 90 CASES**

**DISSERTATION SUBMITTED FOR THE DEGREE OF**

**M.S. GENERAL SUGERY (BRANCH – I)**

**MARCH - 2008**



**THE TAMILNADU  
DR. M.G.R. MEDICAL UNIVERSITY  
CHENNAI, TAMILNADU**

## **BONAFIDE CERTIFICATE**

This is to certify that the dissertation entitled “**A STUDY ON NON-TRAUMATIC ULCERS OF LOWER LIMB**” is bonafide record work done by **Dr.S.PANCHALI** under my direct supervision and guidance, submitted to the Tamil Nadu Dr. M.G.R. Medical University in partial fulfillment of University regulation for M.S. General Surgery, Branch I.

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## **DECLARATION**

I **Dr.S.PANCHALI** solemnly declare that the dissertation titled  
**“A STUDY ON NON-TRAUMATIC ULCERS OF LOWER LIMB”**  
has been prepared by me. I also declare that this bonafide work or a part of  
this work was not submitted by me or any other for any award, degree,  
diploma to any other University board either in India or abroad.

This is submitted to The Tamilnadu Dr. M. G. R. Medical University,  
Chennai in partial fulfillment of the rules and regulation for the award of  
M.S.(General Surgery) Branch – I to be held in March 2008.

**Place :**     Madurai

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**Date :**

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## **INTRODUCTION**

The ulcers of lower limbs constitute one of the common surgical problems with varied etiology, disease manifestation and treatment. The topic of leg ulcers was looked upon as an inferior branch of surgery in the past but due to the modern investigation modalities and treatment facilities, it has changed into an attractive field of surgery with challenging tasks. Through history, detailed clinical examination and non-invasive testing are often required for proper diagnosis, correct treatment and fruitful outcome.

We should strongly endorse a national initiative, which should parallel the advances and understanding of pathophysiology and treatment so as to bring leg ulcers and their management out of the middle ages.

## HISTORICAL REVIEW

- ❖ Ulcers of the lower extremity is an old disease described as early as 1500 BC in the ancient **Egyptian papyrus**.
- ❖ Several causes for ulceration of leg have been put forward even from stone age period ranging from Bad evil, Black bile to Honey like urine in diabetes.
- ❖ The term DIABETES meaning “to pass through” was first coined by Aretaeus in second century AD.
- ❖ The sweet taste of diabetic urine was noted in fifth-sixth century AD by Indian physician Susruta and called it as “madhu meha”.
- ❖ The term **DIABETES MELLITUS** was coined by Jhon Rollo in eighteenth century.
- ❖ **Claude Bernad** discovered the metabolism of Diabetes in nineteenth century.
- ❖ **Insulin** was discovered at the University of Toronto in 1921 by **Fredrick G Banting**, a surgeon and Charles H Best, a student assistant.

❖ Several descriptions of *amputations*, dates back to Hippocrates.

The technique was popularized by **Ambrose pare** -1575.

❖ Young and Lowdhan in 1679 developed flap method of amputation.

❖ The cause and treatment of leg ulcers was evaluated by Anning in 1954- Churchill, London.

❖ The role of varicose veins in venous ulceration was described by **Nicolaides and Miles**

❖ **Thromboangitis obliterans**, a common disease in smokers, causing multiple small vessel occlusions was described by Leo Buerger in 1908.

❖ The terms **Embolism and Deep vein thrombosis** were described by **Rudolf Virchow** in 1854.

❖ The patten of circulation was described by **William Harvey** in 1628.

❖ Recent advances include: Balloon angioplasty for arterial occlusion, Valve repair for venous disorder. Growth factor for rapid wound healing.



## **AIM OF THE STUDY**

1. To study the epidemiology of non-traumatic ulcers of lower limb.
2. To study the commonest organism causing infection & sensitive drug in patients with diabetic ulcers of lower limb.
3. To study number of patients showing osteomyelitis in X-rays.
4. To study percentage of patients undergoing amputation for chronic non-traumatic ulcers of lower limb.

## **METHODS AND MATERIALS**

The clinical material for this study consisted of 90 cases, of chronic non-traumatic lower limb ulcers patients admitted in surgery ward of Govt. Rajaji Hospital during 2005-2007.

The patients were selected with following criteria.

1. Only Non-traumatic ulcer patients are selected
2. Thermal burns scalds are excluded.
3. Superficial infections of skin are excluded.
4. Patients with Hansen's disease leading to trophic ulcer are excluded.

Of the 90 patients included in the study, the relevant data were collected as follows and recorded.

In the proforma, age of patient, sex, occupation, complaints and history is details were obtained with information about Diabetes, HT, TB IHD in past. Personal history about smoking, STD were enquired family-history about connective tissue disorder, congenital hemolytic anaemia were enquired.

Patients were examined in detail about the general condition like anaemia, jaundice, xanthelesma and blood pressure, peripheral pulses.

The affected part with the ulcer was examined in detail for all the features of an ulcer with special concern about side of perforator incompetence and deep vein thrombosis in venous ulcer and motor and sensory testing in patients with diabetic and trophic ulcer.

**The following investigations were carried out as per the need of the study:**

**Urine** : albumin, Sugar

**Blood** : Hemoglobin, total count, differential count, peripheral Smear and ESR

**Blood** : Urea, Sugar, VDRL

**Serum** : Creatinine, cholesterol

**Pus culture and sensitivity** in diabetic ulcer

**Color Doppler study** done in patients with vascular ulcers namely arterial and venous ulcers.

**X-ray** of the part affected to look for osteomyelitis of underlying bone.

**Edge biopsy** in suspected malignant ulcer,

The mode of treatment given whether conservative or surgical, complication, and follow up were recorded in the proforma. Emphasis on level and the type of amputation performed was observed.

All the findings were recorded in the proforma

## REVIEW OF LITERATURE

Chronic ulcers of lower extremity represent a formidable clinical challenge. Successful treatment requires a thorough understanding of the pathologic process, surgical debridement of devitalized tissue and updating various modalities of treatment. Failure to expeditiously recognize the cause, pathology and associated infectious process may lead to devastating consequences, including limb sepsis, amputation and death. *Loudon* in 1805 had quoted that “*leg ulcers are generally looked upon as an inferior branch of practice, an unpleasant and inglorious task where much labour must be bestowed and little honor gained*”.

## CLASSIFICATION OF ULCERS OF LOWER LIMB

### 1. Clinical classification:-

#### **a. Spreading ulcer:**

Surrounding skin of the ulcer is inflamed and floor is covered with profuse and offensive slough without evidence of granulation tissue.

**b. Healing ulcer:**

Floor of the ulcer is covered with healthy granulation tissue has slopping edge, reddish with granulation, and margin is bluish with serous discharge.

**c. Callous or chronic ulcer:**

Ulcer shows no tendency towards healing, has pale granulation tissue and indurated base.

**II. Pathological classification:**

**a. Non- Specific ulcers:**

**i) Traumatic ulcer**

Mechanical : eg: Dental ulcer from jagged tooth

Physical : eg: Electrical or x ray burns

Chemical : eg: caustics

ii) Arterial ulcer

iii) Venous ulcer

iv) Neurogenic ulcer (Trophic ulcer)

v) Cryopathic ulcers

Due to systemic disease –Anemia, diabetes, Rheumatoid arthritis, gout, Martorells ulcer, Bazin's ulcer

**b. Specific Ulcer:** Tuberculosis, Syphilitic, actinomycosis,  
Tropical ulcer, Meleney's ulcer

**c. Malignant ulcer:** Eg: Epithelioma, Rodent ulcer, Malignant  
melanoma

**Arterial ulcer:**

The arterial ulcers are due to peripheral arterial disease, most commonly due to Atherosclerosis (90%), followed by Buerger's disease or Thromboangitis obliterans, Thromboembolic occlusion of arteries and arteritis. The arterial ulcers are due to poor peripheral circulation leading to deficit of nutrients and oxygen, resulting in devitalisation and ulcer formation which is often seen in older patients, in the anterior and outer aspect of the leg, dorsum of the foot, on the toes or on the heel, the parts which are exposed to repeated trivial trauma.

The ulcer is generally accompanied by claudication, ischemic pain, the site of which is dictated by the level of arterial tree occlusion in the lower limb. The arterial anatomy of lower limb shows that the axial artery is the common femoral artery, which continues as superficial femoral artery and popliteal artery. The popliteal artery

divides into posterior tibial and anterior tibial artery. The anterior tibial artery continues as dorsalis pedis artery and the posterior tibial artery give rise to medial and lateral plantar arteries. There is rich communication in the thigh and around the knee joint and scarce below the knee joint. Occlusion of arteries in areas with poor anastomosis gives rise to claudication pain.

Buttock claudication in aorto iliac, thigh claudication in external iliac, calf claudication in popliteal and foot claudication in case of posterior tibial artery occlusion.

The arterial ulcer tend to occur below the medial malleoli with punched out edges, with destruction of deep fascia with exposure of tendons, bone or underlying joint with minimal granulation tissue in the floor. Chronic ischemic changes like pallor, paraesthesia, pulselessness, poikilothermia, paralysis, pre gangrenous changes like dry skin, loss of hair, fissuring of nails occur.

### **Venous ulcer:**

The pathophysiology of venous ulcer is best understood if the anatomy and mechanism of the venous system of lower limb is understood.

## **The venous system of lower limb consists of**

### **i) Deep System:-**

Is constituted by Anterior Tibial Vein, Posterior Tibial Vein.

Popliteal Vein and Femoral Vein.

### **ii) Superficial System:-**

Great (long) saphenous vein

Short saphenous vein.

### **iii) Communicationg system (Perforators)**

Direct

Indirect

Since the blood has to ascend up against gravity, it is aided by the presence of multiple valves in the veins and pumping due to contraction of **gastrosoleus muscle** called **the peripheral heart**.

Normally 90% of the blood is drained by deep system and only 10% by the superficial system. The perforators allow passage of blood from the superficial system to the deep system. If they communicate the superficial system directly to the deep system they are called as ***direct perforators*** and if via muscular veins they are called as indirect perforators.



The medial leg perforators are located on the medial aspect of lower limb, communicating the great saphenous system with the deep system and lateral leg perforators on the lateral aspect communicating the short saphenous system to the deep system.

There are numerous perforators of which the constant are above ankle perforator or *kuster mayo* perforator, leg perforator or *Cockett perforator*, below knee perforator or **Boyd** perforator and adductor canal perforator or **Dodd** perforator. When the valves are damaged following recanalisation after deep vein thrombosis, are absent or are deformed congenitally, it leads to the transmission of blood in the deep system to the superficial system via the perforator leading to venous hypertension, varicosity and ulcer formation. Most often the venous ulcer develops in the lower 1/3 of leg above the medial malleoli due to the presence of constant perforator, high pressure, lack of subcutaneous fat and the area being prone for repeated trauma.

### **Theories of venous ulcer formation**

- 1) **Fibrin cuff theory:** In the presence of venous hypertension a perivascular cuff is formed around capillaries from the exudated

proteins, which acts as a barrier to diffusion, preventing nutrition exchange, leading to tissue damage and ulceration.

**2) White cell trapping theory:** There is sequestration of leukocytes in the microcirculation, leading to release of proteolytic enzymes, causing injury and ulceration.

### **Characteristics of Venous ulcer**

90% of the ulcers are typically situated on the medial aspect of the lower third of leg above the medial malleoli called the ***gaiter area*** often associated with varicosity of veins, hence the term venous ulcer. They are painless with surrounding eczema and pigmentation with sloping edges and are always superficial to deep fascia.

Pitting oedema of the legs is initially present at the end of the day and later becomes persistent and non-pitting. The skin may have “lipodermatosclerosis” which usually starts around the ankle, but can involve the entire lower leg resulting in an “**inverted champagne bottle**” or “**inverted piano leg**” appearance.

**Clinical classification of chronic lower extremity venous disease.**

Class 0 - No visible or palpable signs of venous disease

Class 1 - Teleangiectasia, reticular veins, malleolar flare

Class 2 - Varicose veins

Class 3 - Varicosity with dependent edema

Class 4 - Skin changes ascribed to venous disease

Eg pigmentation, eczema, lipodermatosclerosis.

Class 5 - Skin changes as defined above with healed  
ulceration

Class 6 - Skin changes as defined above with active ulceration

### **Difference between arterial and venous ulcer.**

<b>Character</b>	<b>Arterial Ulcer</b>	<b>Venous Ulcer</b>
Site	Dorsum of foot, toes, heel usually below medial malleoli	Lower 1/3 of leg just above the medial malleoli
Pain	Present	Often absent
Edge	Punched out	Sloping
Depth	Tendon and bone exposed	Superficial to deep fascia
Surrounding area	Pre gangrenous change	Eczema, Pigmentation (lipodermatosclerosis)
Vascular disorder	Absent pulse	Past H/o DVT, Varicosity of veins
Bleeding	Absent	Present

### **Trophic ulcer (Neurogenic ulcer)**

These ulcers develop as a result of repeated trauma to insensitive part of the body. Neurological disturbance in the form of sensory loss is the cause behind the ulcer formation. These ulcers are commonly seen on the heel of the foot when the patient is ambulant and on the back of heel and buttock when the patient is bed ridden. The ulcer has punched out edge with slough in the floor with tendon and even bone being exposed. The base is indurated, surrounding skin has no sensation. The causes may be spinal cord injury, Hansens disease, peripheral neuropathy, and diabetes.

### **Cryopathic ulcer.**

It results from the exposure to intense cold. Chilblains result from exposure to chill weather that results in blister formation and ulceration. Frost bite occurs when any part of the body is exposed to wet cold below freezing point, which causes arteriolar spasm, freezing of tissues and denaturation leading to gangrene and full thickness loss of skin.

### **Diabetic ulcer:-**

Three factors play a predominant role in the genesis of diabetic ulcer.

- 1) Diabetic Neuropathy
- 2) Diabetic atherosclerosis resulting in ischemia
- 3) Hyperglycemia.

The neuropathic factor impairs sensation and thus favors the neglect of minor injuries and infections. So inflammation and damage to the tissues are ignored. Thick callosities develop on the sole and provide means for the entry of organisms.

## **Theories for Diabetic Neuropathy :**

### **1. Metabolic theory**

The increased blood glucose level leads to increased formation of sorbitol, which is found to be toxic to nerves.

Reduction in the phosphoinositol leads to reduced myelin production

### **2. Vascular theory**

This theory suggests the neuropathy is due to microangiopathy leading to reduced blood flow to the nerve sheath bundles leading to chronic ischemia and progressive nerve damage.

Diabetes is associated with microangiopathy as well as macroangiopathy since it hastens atherosclerosis.

Excess of sugar in the tissues favors growth of organism and lowers resistance to infection. Osteomyelitic changes can occur in the bone due to spreading infection and bone necrosis.

The ulcers are characterized by rapid progression and they spread along all the tissue planes damaging fascia, tendons, muscle and bones often with mild local and general symptoms

**Grading of diabetic foot ulcers are:**

- Grade 0 : Preulcerative lesion
- Grade I : Superficial Ulcer.
- Grade II : Deep ulcer with no tendon or bone involvement
- Grade III : Deep ulcer extending to tendon or bone
- Grade IV : Deep ulcer with Gangrene
- Grade V : Infected Diabetic ulcer

**Anemia:-**

Congenital hemolytic anemias like spherocytosis, sickle cell anemia predispose to ulcer formation. Sickle cell anemia predisposes to ulcer formation due to occlusion at micro vascular level. Stasis and hypoxia predispose to occlusion of vessels by sickling leading to infarction and ulcer formation. It is associated with jaundice and gall stone disease due to chronic haemolysis with positive family history. The ulcer is surrounded by black pigmentation.

**Martorell's ulcer (hypertensive ulcer)**

Occurs in older patients with long standing hypertension with atherosclerosis. A local patch of skin on the back or outer side of calf suddenly necroses and sloughs away leaving a punched out ulcer

extending down to the deep fascia, with very severe pain. This condition may be bilateral and the underlying pathology is sudden obliteration of the end arteries of the skin of this region, which is already having sparse arterial supply from atherosclerosis. All the peripheral pulses are felt normally. It takes months to heal.

### **Erythrocyanoid ulcer (Bazin's disease)**

It is an exclusive disease of young women with abnormal amount of subcutaneous fat, with thick ankles combined with abnormally poor arterial supply. In these patients the perforating arteries arising from the Posterior Tibial Artery and peroneal arteries supplying the lower third of leg and ankle are abnormally small and even absent. The ankle skin is abnormally sensitive to temperature changes. When the weather is cold the ankle is blue, cold and often tender. In hot weather chronic reactive hyperaemia leads to hot edematous swollen and painful ankle. There are multiple small painful nodules, which break down to form ulcers, which are small and multiple.

### **Ulcers due to other systemic disease:**

Connective tissue disorders, gout, leukemia, Paget's disease, ulcerative colitis can rarely give rise to ulcers of lower limb.



### **Ulcers due to specific infections:**

- 1) Tropical ulcer
- 2) Tuberculous ulcer
- 3) Syphilitic ulcer
- 4) Meleney's ulcer
- 5) Yaw's
- 6) Maduramycosis

### **Tropical ulcer:-**

It is a rare ulcer, which occurs on the legs and feet following trauma or insect bite. It is caused by Vincent's organism (*Fusobacterium* and *Borrelia vincenti*). It commences as a papulopustule which bursts in a couple of days to form a painful, foul smelling ulcer. The edge of this ulcer is typically undermined due to considerable infiltration in the surrounding skin. The floor is covered with copious seropurulent discharge. The spreading process ceases after a few weeks resulting in a non-healing ulcer which refuses to heal for even months. In few cases it assumes phagedenic (eating) characteristic resulting in wide spread destruction of the soft tissue necessitating even an amputation. Rarely

squamous carcinoma may supervene. After a long period of healing it leaves behind a parchment paper like pigmented scar.

### **Tuberculous ulcer:**

This most commonly results from bursting of caseous lymph node, but can also develop when cold abscess from bone and joint tuberculosis breaks out on the surface. The ulcer is irregular in shape and has a undermined thin edge. The floor is covered with pale granulation tissue (apple jelly appearance) with thin serosanguinous discharge. The base is not indurated; the surrounding skin shows bluish black pigmentation. The regional nodes are enlarged non-tender, firm and matted; there may be systemic evidence of tuberculosis of lung or other part of the body.

### **Lupus Vulgaris:**

It is a form of cutaneous tuberculosis occurring in young adults, which starts as cutaneous nodule which gradually turns into ulcer. The ulcer remains active in periphery and spreads outwards, whereas in the center they gradually heal. Due to its destructive nature it is called 'Lupus' which means 'wolf'. Squamous cell carcinoma may grow from the scar of Lupus vulgaris to form Marjolin's ulcer.

**Syphilitic gummatous ulcer:-**

It occurs in late syphilis and is due to inflammatory reaction in perivascular lymphatics of the terminal vessels with subsequent obliterative endarteritis, necrosis and fibrosis.

It usually occurs in adults as a single, slowly progressive ulcer. Pain and tenderness are absent. Commonly occurs over the tibia, with punched out indolent edge and yellowish grey gummatous tissue (wash-leather slough) in the floor. Lymph nodes are not involved, unless secondarily infected.

**Meleney's ulcer:-**

It is due to symbiotic infection of micro-aerophilic non-hemolytic streptococci and staphylococcus aureus. It occurs de novo in association with ulcerative colitis or as a complication of a previously existing ulcer. It has undermined edges and foul smelling abundant granulation tissue with copious seropurulent discharge; it is surrounded by deep purple zone, which in turn is surrounded by outer zone of erythema. These ulcers are very painful, tender, show tendency to spread and make the general condition of the patient to deteriorate if not treated.

**Yaws:-(Framboesia)**

It is caused by *Treponema pertenue*. The ulcers can be found on the leg or foot as abrasions. It is painless and heals in few weeks with paper like scar.

**Maduramycosis (Mycetoma foot/Madura foot)**

It is a chronic infective granulomatous disease caused by a group of fungi occurring usually in tropical countries. In India it commonly occurs in Madurai, Chennai, Jothpur and Bikaner. It is caused by *Nocardia*, *Madura mycetomi* and *Streptomyces madurae*. The fungal organism gains entry through a thorn prick in bare foot walkers. It characteristically presents with triad of 3S'-Swelling, Sinus and Sero Sanguineous granular discharge. The granules may be yellow or red(*Nocardia* group), black or white (*Madurella mycetomi*). There are multiple discharging sinuses with pigmentation and induration of skin. The regional nodes are not involved. Diagnosis is confirmed by microscopic examination of granules, which show ray or fan like arrangements and hence called as 'ray fungus', which are gram-positive branching filaments.

## **MALIGNANT ULCERS**

### **Squamous cell carcinoma (Epidermoid carcinoma/epithelioma)**

It arises from the prickle cell layer of the skin. It is mostly seen after 40 years of age. It begins as a small nodule, which enlarges, and gradually the center becomes necrotic and sloughs out and thus ulcer develops. Such an ulcer is oval or circular in shape, but size varies considerably. The edge of the ulcer is raised and everted, floor is covered by necrotic tumor tissue, serum and blood. Base of the ulcer is indurated which is the pathognomonic sign of epithelioma. In later cases it becomes fixed due to involvement of the deeper structures regional lymph nodes are almost always enlarged be it due to metastasis or secondary infection. Metastatic node is hard and fixed.

### **Basal cell carcinoma (Rodent ulcer)**

It originates from the basal layer of rete Malpighi of the skin. It is a low-grade malignancy most commonly seen in white-skinned people, often involving the sun-exposed area namely the face.

It starts as a small brownish-red nodule with translucent color and shiny surface showing a network of capillaries. At this stage it is diagnosed due to its hardness, painlessness and presence of capillaries,

later the tumor ulcerates with a well-defined hard and raised edge with beaded appearance. It infiltrates into the surrounding as well as deeper tissues, even up to the bone and hence the name Rodent. At first it may itch, but at a later stage it may be painful if it has involved any nerve.

Dissemination by lymphatic or blood vessels does not occur, so that regional nodes are not enlarged.

### **Malignant melanoma:**

It is a malignant tumor of melanocytes which originate from the neural crest and so ectodermal in origin. It may occur de novo or most often from benign mole. The following points suggest malignant conversion of a mole

- Asymmetry
- Border irregularity
- Color variation
- Diameter more than 6 mm size
- Elevation

The predisposing factor is exposure to sunlight, functional and dysplastic nevi, immunosuppression Occurs commonly in females and occurs between 30-50 years.

The size and appearance vary clinically because it has vertical and radical growth. The depth of invasion can be measured by Clark's level of invasion or by Breslow's index. It can have satellite nodules and in transit metastasis due to lymphatic spread. Regional nodes and visceral metastasis to liver, lung, skin, gut and bone are common.

### **Marjolin's ulcer:-**

This is a squamous cell carcinoma arising from a long-standing benign ulcer or scar. The commonest ulcer to become malignant is a long standing venous ulcer and the scar in a old scar of a burn. It is slow growing and is less malignant. Its edges are not raised or everted. It is absolutely painless and lymphatic metastasis is unusual as the nerves and lymphatics are already destroyed or occluded by previous chronic lesion of skin. Unlike squamous cell carcinoma it is radioresistant.

### **Kaposi's sarcoma:-**

It is a slow growing malignant mesenchymal tumor, a rare malignant cause of leg ulcer. It is normally seen in Jews, Italians and eastern Europeans. Males in the middle of their lives are prone. Multiple painless plum colored nodules arise, which may ulcerate and

present as an ulcer. Enlarged lymph nodes and hepatosplenomegaly are the characteristic features. It is associated with hemolytic anemia, lymphoma, diabetes and AIDS.

### **Management of lower ulcers:**

#### **Investigations:**

Chronic ulceration of lower limb constitutes one of the major health problem and throws enormous burden on the health service in terms of dressing, cost of investigation and staff time.

For proper management of lower limb ulcers the most important thing is thorough clinical examination and few special investigations for confirmations, which are stream lined according to the history and clinical findings.

#### **Special tests are done as follows**

- **Urine analysis** – If diabetes is suspected
- **Full blood examination**

Complete haemogram is done if anemia is suspected as the cause for the ulcer. Peripheral smear shows spherocytes or sickle cells in congenital hemolytic anemia. Further confirmation is done by osmotic fragility test for spherocytosis using various concentration of NaCl solution, for sickle cell anemia using sodium metabisulphide.



### **Blood sugar and renal parameters:-**

All patients with ulcers of lower limb must undergo screening for Diabetes mellitus with urine sugar; random blood sugar and glucose tolerance test if necessary. Renal parameter should be evaluated since renal failure can co-exist with diabetes and renal failure by itself may delay ulcer healing.

- **Lipid profile**
- **Culture and smear of ulcer discharge**

The bacteriology of infective organism causing infection in leg ulcer has to be identified for its effective control and fruitful outcome of the treatment. The type of organism causing infection depends on the cause of the ulcer. In Diabetic foot, the common organisms are Staphylococcus aureus, Enterococci, gram negative bacilli like E.Coli, Proteus, Klebsiella, Pseudomonas and some anaerobes like Peptococcus, Bacteroides, Clostridium etc. In ulcers due to vascular disorders, infections are often caused by Staphylococcus and Streptococcus.

The studies show that *Proteus* and *Peptococcus* rank first among *aerobes* and *anaerobes* respectively. In most of the cases mixed

infections occur and hence combination therapy with Gentamycin, Ciprofloxacin or Ceftazidime with either Metronidazole or Clindamycin is appropriate.

- **Edge biopsy of ulcer:**

Edge Biopsy is taken from the ulcer if malignancy or infection due to specific organism e.g. Tuberculosis is suspected. In malignant ulcer biopsy shows typical epithelial pearls pattern in squamous cell carcinoma, palisading manner of arrangement of cells in basal cell carcinoma and nests of melanin containing cells, with invasion into dermis and deeper tissues, with atypical morphological appearance and increased mitotic figures in malignant melanoma.

- **Ankle Doppler pressure and arteriography:**

It is done for peripheral arterial disease. Ankle blood pressure and brachial blood pressure are measured by using Doppler probe and ankle brachial index is calculated as follows.

$$\text{ABI} = \text{ankle blood pressure} / \text{Brachial blood pressure}$$

Normal value is  $1 \pm 0.1$ .

Reduction in ABI shows the degree of occlusion.

Arteriography is indicated only if definitive vascular procedures are planned. It shows the site of occlusion of artery and corkscrew pattern of collaterals in chronic ischemia. It is done by Schioldinger's technique.

- **Duplex Scanning with color Doppler**

The advent of colour-flow duplex scanning has revolutionized the assessment of peripheral vascular disease.

1. Patency & competence of the deep veins
2. Site & level of major incompetence involved
3. Anatomy of popliteal fossa identified
4. duration & velocity of venous reflex

In patients with suspected DVT, presence of thrombus can be seen.

- **Digital sub-traction Angiography:**

more advanced method of arteriography in which computer subtract the pixels of a first image of the series from subsequent images removing extraneous background and so provides great clarity.

➤ **Transcutaneous oximetry:**

Transcutaneous measurement of O<sub>2</sub> tension for assessing tissue perfusion has a role in assessment of critical ischemia particularly in Diabetic patients with extensive vascular calcification

Normal tcpO<sub>2</sub> level in foot - 50 - 60 mm Hg

> 40 mm Hg – Predictive for healing of foot lesions

< 10 mm hg - failure to heal

Measuring the tcpO<sub>2</sub> is particularly useful in determining the level of amputation.

➤ **Nerve conduction velocity studies (NCV)**

For neuropathic ulcer, the conduction velocity of a nerve is calculated after application of electric stimuli, by measuring the time taken by the action potential created by the stimuli to travel along the nerve. Nerve conduction velocity is useful in detecting sensory and mixed nerve involvement.

➤ **Screening for Autoimmune diseases**

When connective tissue disease like Rheumatoid arthritis, SLE is suspected, Rose Waller Test and Anti nuclear antibody testing are done.

After confirmation of the cause of lower limb ulcer, the treatment is carried out as follows:

## **TREATMENT**

### **➤ CURRENT METHODOLOGY IN CHRONIC WOUND MANAGEMENT**

#### **A. DRESSINGS**

- a. Alginates
- b. Hydrocolloids
- c. Hydrogels
- d. Films
- e. Foams
- f. Medicated dressings

#### **B. PHYSICAL THERAPIES**

- a. Hyperbaric Oxygen
- b. Lasers
- c. Magnetic stimulation
- d. Ultrasound stimulation
- e. Vacuum devices
- f. Warming

#### **C. BIOLOGICAL THERAPIES**

- a. Cytokines
- b. Growth factors
- c. Protease inhibitors
- d. Cadaver skin
- e. Epidermal allograft
- f. Dermal allograft

## **SURGICAL INTERVENTIONS**

- g. Debridement
- h. Drainage
- i. Excision
- j. Skin grafting
- k. Revascularisation
- l. Reconstruction
- m. Amputation

The current concept of chronic wound management is multidisciplinary approach. The general surgeon can give support for wound debridement and delayed suture; split thickness grafts or flap covers may require plastic surgeons help. A vascular surgeon provides help with revascularization, sometimes with the help of interventional radiology, and to undertake venous surgery when indicated. All this is supported by endocrinologists (diabetic ulcers), care of the elderly by physicians, nurse specialists and professions allied to medicine, such as podiatrists. Thus chronic wound management requires professional expertise from a wide range of backgrounds.

## **Arterial ulcers:**

General measures like absolute abstinence from smoking, regular use of analgesics, haemorheological agents like Pentoxifylline, Dextran -40, Antiplatelet agents, vasodilators like calcium channel blockers, PGE<sub>2</sub>, prostacyclin, antidepressants especially tricyclic antidepressants, simple dressing and removal of gangrenous part has to be carried out.

The presence of ulcers usually means that there is severe degree of ischemia and hence local treatment of the ulcer is unlikely to be effective unless the arterial supply can be improved either by

- Direct arterial surgery
- Ballon angioplasty
- By-pass operation
- Lumbar sympathectomy
- End arterectomy

Direct arterial surgery is attempted in the pathology of large and medium sized vessels. Before arterial surgery is undertaken arteriography is a must.

**Lumbar sympathectomy** increases the flow of blood to the extremity by peripheral vasodilatation of arterioles in the skin bed. This may be sufficient to heal small superficial ulcers or relieve ischaemic rest pain in those with severe distal disease.

Chemical lumbar sympathectomy could be performed by using a translumbar approach with image intensifier with alcohol or phenol.

### **Venous ulcer:**

Treatment of venous ulcer is either conservative or operative.

### **Conservative (Bisgaard's) line of management:**

- The foot end of bed elevated about 60 cm
- Four layered dressing which can be left for up to a week if there is not much discharge.
- Compression bandaging or hosiery with pressure upto 30-45 mm of Hg – '**4 layer bandage**' developed at Charing Cross Hospital, London. It achieves pressure of 45 mm Hg and produce healing of 70% of venous ulcers within 12 weeks, must be changed once or twice a week.
- Antibiotics and desloughing agents are not indicated unless the ulcers are severely infected.



- Correct any associated general disorders like obesity, anemia, vitamin deficiency.
- Better avoid local applications as they are unnecessary and causes sensitization
- To prevent recurrence wear supportive stockings.

**The conservative method is very effective in**

- 1) Ulcer less than 5cm size.
- 2) With No associated deep vein malfunction.
- 3) Ambulant patient
- 4) No Lipodermatosclerosis.
- 5) No associated arterial disease.

If the ulcer fails to heal, the associated arterial diseases or malignant conversion of the ulcer (Marjolin's) should be excluded.

**Operative management:**

**Indications:**

Indications for operative management are failure of conservative treatment. Presence of extensive ulcer (more than 5 cm size) that would require prolonged conservative treatment before healing occurs.

## **The surgical options for venous ulcer management :**

### **1.Ligation of Incompetent site-**

- a) Trendelenburg operation for saphenofemoral junction
- b) Dodd and Crockett operation for perforator incompetence.

### **2. Open Perforator Ligation (Linton's Procedure) – Direct ligation through a longitudinal incision**

### **3. Newer Options:**

- a) Direct valvuloplasty of kistner
- b) By pass surgery for major vein occlusion. Eg. Palma operation
- c) High dose pentoxifylline 800 mg. tid has shown to accelerate the healing.

### **Sub-fascial endoscopic perforator surgery (SEPS)**

Endoscopic division of previously marked medial calf perforators using single port technique & application of 2 clips to a perforator. Undoubtedly safe & feasible.

Compared to open Linton operation, with less scarring & much greater tendency towards fast recovery.

## **Newer options:**

### **Gene therapy in peripheral vascular disease**

Isner et al. has tried gene therapy for improvement of vascular supply of lower limbs.

A vascular endothelial growth factor (VEGF) is a secreted endothelial cell mitogen that promotes angiogenesis. It is still at an experimental stage.

## **Trophic ulcer:**

### **A) Treatment of the cause:**

The prime cause predisposing to trophic ulcer formation has to be removed viz-avoiding pressure points, bare foot walking; and alcoholism, correcting diabetes mellitus and vitamin deficiency.

### **B) Treatment of the limb:**

Clawing of the toes due to intrinsic muscle paralysis is treated with a well fitting shoes and inner sole. When the toes become flexed, amputation may be necessary.

### **C)Treatment of the ulcer:**

Trophic ulcers are best treated by rest, simple dry dressing, chiropody, soft shoes, thick socks and sponge rubber inner soles. Persistent trophic ulcer will require excision of the ulcer with closure of defect by primary suture, skin graft, local flap cover or distant free flaps.

### **Cryopathic ulcer :**

- i) Protection from cold and warming of whole body
- ii) Vasodilator drugs may be tried in severe and recurrent cases
- iii) Regular dressing and oral antibiotics and analgesics.
- iv) Excision of dead tissue and skin grafting
- v) Hyperbaric oxygen
- vi) Para vertebral injection of sympathetic chain
- vii) Amputation

### **Diabetic ulcers:**

#### **Prevention:**

The diabetic patients foot are an “AT RISK FOOT” to get ulcerated and the treatment of it causes loss of lot of economy of a country and hospital source and hence meticulous measures should be

considered for its prevention. Patients should be advised about foot care, avoiding bare foot walking, to check for foreign bodies and nails in foot wear, to wear individually designed and well fitting foot wear, chiropody services, avoiding warmth application, dietary modification and diabetic control with regular medical surveillance in well established diabetic department.

## **TREATMENT**

1. **Control of infection:** control of infection is the first priority in the management of Diabetic ulcers. If there are signs of spreading infection or systemic involvement (i.e. fever, tachycardia or loss of diabetic control) the patients should be admitted in hospital for intensive treatment with parenteral antibiotics. The usual infecting organism is Staphylococcus aureus, Gram negative bacilli and mixed infections.
2. **control of Blood glucose:** Control of blood glucose need temporary treatment with plain insulin. With plain insulin the control of blood glucose is easier and has less been complications.

**3. Surgical role:** The surgical role may involve desloughing of ulcer, drainage of pus in the tissue planes and minor or even major amputations. The key to success is to remove all dead tissue and to leave the wound open. Amputations are done for removal of dead necrotic tissues, uncontrolled infection with wet gangrene, associated atherosclerotic changes causing dry gangrene and uncontrolled osteomyelitis with soft tissue necrosis. Amputations can be performed as an emergency or elective procedure. The level of amputation is dictated by the extent of tissue damage, infection and vascularity.

The aim of surgery is to allow the patient to become ambulant. The raw area left behind after the control of infection can be skin grafted or covered with flap depending on the site and size.

**Ulcers due to systemic diseases and specific infections:**

Systemic conditions predisposing on ulcer formation namely anemia, connective tissue disorders, and gout have to be identified and managed medically. Surgical role comes to play for complications and non-responding ulcers. Steroids are effective in autoimmune disorders. Allopurinol is effective in gout.

Ulcers due to infective etiology have to be managed according to causative organism with antibiotics similar to that of any other site.

**Malignant ulcers:**

In management of malignant ulcers, surgery, radiotherapy, chemotherapy and other options available has to be considered. The stage of the disease, type of malignancy, grade and site dictates the mode of management.

For the primary site wide excision with 2-5cm clearance with skin grafting is indicated in squamous cell carcinoma and malignant melanoma. In basal cell carcinoma wide excision with 2 cm clearance is the best treatment though radiotherapy cryotherapy, mohs microsurgery can be adopted.

In case of marjolins ulcer wide excision is the best option. In Kaposi sarcoma with one or two large lesions, excision with margin or RT and multiple chemotherapy with vinblastin and immunotherapy are ideal.

For the regional nodes in case of squamous cell carcinoma and malignant melanoma block dissection for mobile nodes and irradiation with palliative chemotherapy for fixed nodes is adopted.

For in transit metastasis and recurrent melanoma isolated lower limb perfusion therapy can be given. Lower limb malignant ulcer with distant metastasis is managed with chemotherapy or radiotherapy according to the site and responsiveness to each therapy.



## OBSERVATION

### AGE WISE INCIDENCE :

Age Group	No.of patients	Percentage
Upto 20 years	10	11.11%
21-40 years	18	20.00 %
Above 40 years	62	68.89.%
Total	90	100 %

### SEXWISE INCIDENCE :

Type of Ulcer	Male		Female		Total	
	Number	%	Number	%	Number	%
Diabetic	27	30%	11	12.2%	38	42.22%
Venous	18	20%	3	3.3%	21	23.33
Arterial	8	8.8%	2	2.2%	10	11.11%
Malignant	7	7.7%	2	2.2%	9	10%
Trophic	4	4.4%	1	1.1%	5	5.56%
Tuberculous	1	1.1%	1	1.1%	2	2.22%
Tropical	2	2.2%	.	-	2	2.22%
Others	2	2.2%	1	1.1%	3	3.33%
Total	69	76.6%	21	23.3%	90	100%

### INCIDENCE OF VARIOUS LOWER LIMB ULCERS

S.No	Type of ulcers	No.of cases	Percentage
1.	Diabetic ulcer	38	42.22 %
2	Venous ulcer	21	23.33%
3	Arterial ulcer	10	11.11%
4	Malignant ulcer	9	10 %
5	Trophic ulcer	5	5.50 %
6.	Tuberculous ulcer	2	2.22%
7	Tropical ulcer	2	2.22%
8	Others	3	3.33%
	Total	90	100%

## BIOPSY REPORT IN MALIGNANT ULCER

Type of malignancy	No.of patients	Percentage
Squamous cell CA	6	66.67%
Malignant melanoma	2	22.22%
Sarcoma	1	11.11%
Total	9	100%

## INFECTIVE ORGANISM IN DIABETIC ULCER

Organism	No.of patients	Percentage
E. Coli	17	44.74 %
Klebsiella	9	23.68 %
Proteus	5	13.16%
Pseudomonas	2	5.26%
Staphylococcus	2	5.26%
Anaerobes	2	5.26%
Mixed	1	2.63%
Total	38	100%

### SENSITIVE DRUG IN DIABETIC ULCER

S.No.	Sensitive drug	No.of patients	%
1.	Cipro	26	68.42 %
2.	Amikacin	13	34.21%
3.	Gentamicin	16	42.11%
4.	Pencillin	4	10.53%
5.	Cephalosporin	6	15.79%
6.	Metronidazole	2	5.26%

## TYPE OF TREATMENT

S.No.	Type of Ulcer	No.of patients	Treatment		Percentage	
			conservative	Surgical	C	S
1.	Diabetic	38	9	29	10%	32.22%
2.	Venous	21	10	11	11.11%	12.22%
3.	Arterial	10	4	6	4.44%	6.67%
4.	Malignant	9	-	9	-	10%
5.	Trophic	5	-	5	-	5.56
6.	Tuberculous	2	2	-	2.22%	-
7.	Tropical	2	2	-	2.22%	-
8.	Others	3	3	-	3.33%	-
	Total	90	30	60	33%	67 %

### INDICATION and INCIDENCE OF AMPUTATION

Type of Ulcer	Total no. of patients	No.of Amputee	% of Amputee
Diabetic	38	14	15.56 %
Venous	21	-	-
Arterial	10	4	4.44%
Malignant	9	5	5.56%
Trophic	5	1	1.11%
Tuberculous	2	-	-
Tropical	2	-	-
Others	3	1	1.11%
Total	90	25	27.78 %

## LEVEL OF AMPUTATION

Level of amputation	No.of patients	%
Above knee	2	8%
Below Knee	11	44%
Syme's	2	8%
Transmetatarsal	2	8%
Ray amputation	3	12%
Toe disarticulation	5	20%
Total	25	100%



## **Results of the Study :**

- Epidemiology of this study revealed that patients with non-traumatic ulcers of lower limb constitute 0.2% of inpatients admission per year.
- Among the 90 cases studied diabetic ulcer constitute 43% crowns as the commonest cause of lower extremity ulcer in Govt. Rajaji Hospital whereas literature reveal venous ulcer as the commonest type making 70%.
- Ulcers due to vascular disorders and malignancy constitute the next common with 36% and 10% respectively.
- Commonest age group involved is above 40 years (69%) which correlated with other studies.
- In this study male patients occupy the predominantly affected sex with male : female 3:1.
- The venous ulcer which constitute about 24% is common in patients who are agricultural labourers
- Arterial ulcers constitute about 12% are common in chronic smokers revealing that smoking is the commonest predisposing factor for arterial disorders.

- Pus culture and sensitivity done for diabetic patients reveal E.coli as the commonest organism (45%) klebsiella (24%), Proteus 14%, when compared to standard studies, which mention mixed infections, staphylococcus and proteus as common organism.
- The sensitivity of organisms in diabetic ulcer studied shows Ciprofloxacin sensitivity in 69% and sensitive to Amikacin (35%) and gentamicin in 42%.
- 9 out of 90 patients showed osteomyelitic changes of which seven were diabetic emphasizing that diabetic ulcer patients are prone for osteomyelitis of underlying bone.
- Most of the patients with lower limb ulcer need surgical treatment stressing on the importance of proper diagnosis and correct surgical procedure for better outcome.
- About 16% of diabetic ulcer patients underwent amputation, 5% of arterial ulcer patient underwent amputation either minor or major amputation.
- Follow up of patients reveal that patients with diabetic ulcer and venous ulcer are prone for recurrence.

## **CONCLUSION**

Diabetic ulcer constitute the commonest cause of lower extremity ulcers (43%). Ulcers due to vascular disorders and malignancy coming next. Commonest age group involved is above 40 years. Arterial ulcers are common in smokers. Pus-culture and sensitivity done for diabetic ulcer patients reveled E-coli as the commonest organism and ciprofloxacin as the most sensitive drug. 28% of patients with lower extremity ulcer underwent amputation either minor or major amputation.

Multi-layered, elastic graduated compression therapy is the single most effective means of healing venous ulcer.

Prompt and proper diabetic control is essential. Efficient assessment clinics to evaluate the patients and select those who would benefit from intervention are essential to make best use of limited resources and also periodic review for prevention of recurrence and proper management.

## **BIBLIOGRAPHY**

1. A manual on clinical surgery – sixth edition by S. Das.– Exam.  
Of ulcers and different types of leg ulcers
2. Clinical surgery edited by Alfred cuschieri – Fourth edition.  
Exam. Of ulcer.
3. Clinical Dermatology of Thomas P. Halip .Venous ulcer and  
dermatopathy.
4. Dermatology in several medicine by Fitzpatrick II edition
5. Diabetes Mellitus Diagnosis and Treatment – edited by Mayer  
B. Davidson
6. Fitzpatrick's Textbood of Dermatology 5<sup>th</sup> edition Vol II  
Lower limb ulcers
7. Sabison's Text book of Surgery 17<sup>th</sup> edition.
8. Fredrick's Doppler ultrasound principle and instruments  
hospital infection by
9. Hospital Today, Special Journal of Surgery
10. Textbook of Dermatology – Rook and Wilkinson's Sixth  
edition
11. Smith & Nephew Ltd, Leg ulcer guidelines 2006, Jan

12. Text book of Diagnostic Microbiology by Conni R. Mahon II edition.
13. Oxford Text book of Surgery edited by Peter J. Morsis and Ronald A. Malt. Vol I. Lower limb amputation.
14. Stuart Maddin's current dermatology – therapy, lower limb ulcers. Conservative ulcer management.
15. Scientific principles and practice in surgery. Edited by Lazar J. Greenfield and Michael – Lippincote – Diabetic ulcers and leg ulcers.

# PROFORMA

Name

Age/Sex

IPNo.

Unit / Ward

## Occupation

## History

## Leg Ulceration duration

Site

## Pain

## Dilated Veins

## Pigment and Itching

## Bleeding

Discharge

## Claudication

## Sensory Loss

## Deformity

## Past History

## Diabetes

Duration Treatment – Yes / No

## Tuberculosis

## Hypertension

## IHD / Valvular heart disease

Personal H/o

## Smoking

STD

Family H/o

Congenital Hemolytic Anemia

Connective Tissue Disorder

Examination

General

Anemia

Jaundice

Xanthelesma

Fever

Local

Inspection

palpation

Site

Temperature

Size

Tenderness

Shape

Edge

Margin

Base

Floor

Surrounding Area

Surrounding Area

Regional Node

Special Test

1. Venous Ulcer - System involved : GSV / ASV

Incompetent site

DVT

2. Diabetic and Trophic Ulcer - Sensory Testing Motortesting

Light touch	Power
Crude touch	Deformity
Joint sensation	
Vibration sensation	

## INVESTIGATION

Urine	:	Albumin		
		Sugar		
Blood	:	HB %	TC	DC
		Peripheral smear		ESR
Blood	:	Urea	Sugar	VDRL
Serum	:	Creatinine	Cholesterol	
PUS culture and Sensitivity	-	Organism		
		Sensitive Drug – Ci, G A, M, Ce, P		

Biopsy Report of Ulcer :

X ray of local part	Osteomyelitis present / Absent
---------------------	--------------------------------

Colour Doppler Study :

Final Diagnosis

Treatment

Type	Conservative / Surgical
Amputation	Yes / No
	Level / Toe / TM / Symes / BK / AK
	Method guillotine / Flap

Complication :

Follow up :



## ABBREVIATIONS USED IN THE MASTER CHART

A	Amikacin	PL	Phantom limb
Agri	Agricultural labourer	Prot	Proteus
AK	Above knee	Pseud	Pseudomonas
B	Bleeding	Ray	Ray amputation
BK	Below knee	R	Revision
C	Conservative	S	Surgical
Ce	Cephalosporin	SCC	Squamous cell carcinoma
Ci	Ciprofloxacin	STS	Soft tissue saroma
Condu	Conductor	Staph	Staphlococcus
E.coli	Escherichia coli	TB	Tuberculous
F	Flap	TD	Toe disarticulation
G	Gentamicin	Tech	Technical Job
H.W.	House wife	TM	Transmetatarsal
Kleb	Klebsiella	Tt	Treatment
M	Metronidazole	WD	Wound dehiscence
Mm	Malignant melanoma	WI	Wound infection
Off	Officer	WE	Wide excision
P	Pencillin		

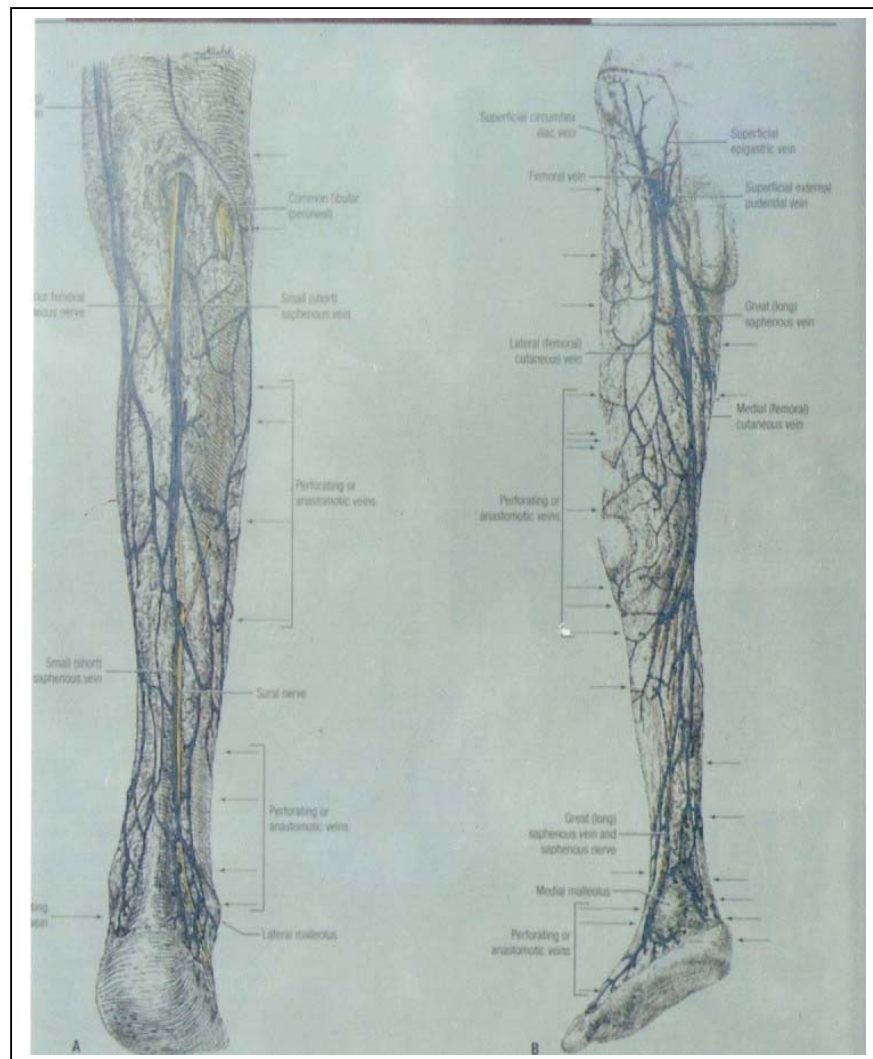
## VENOUS ULCER



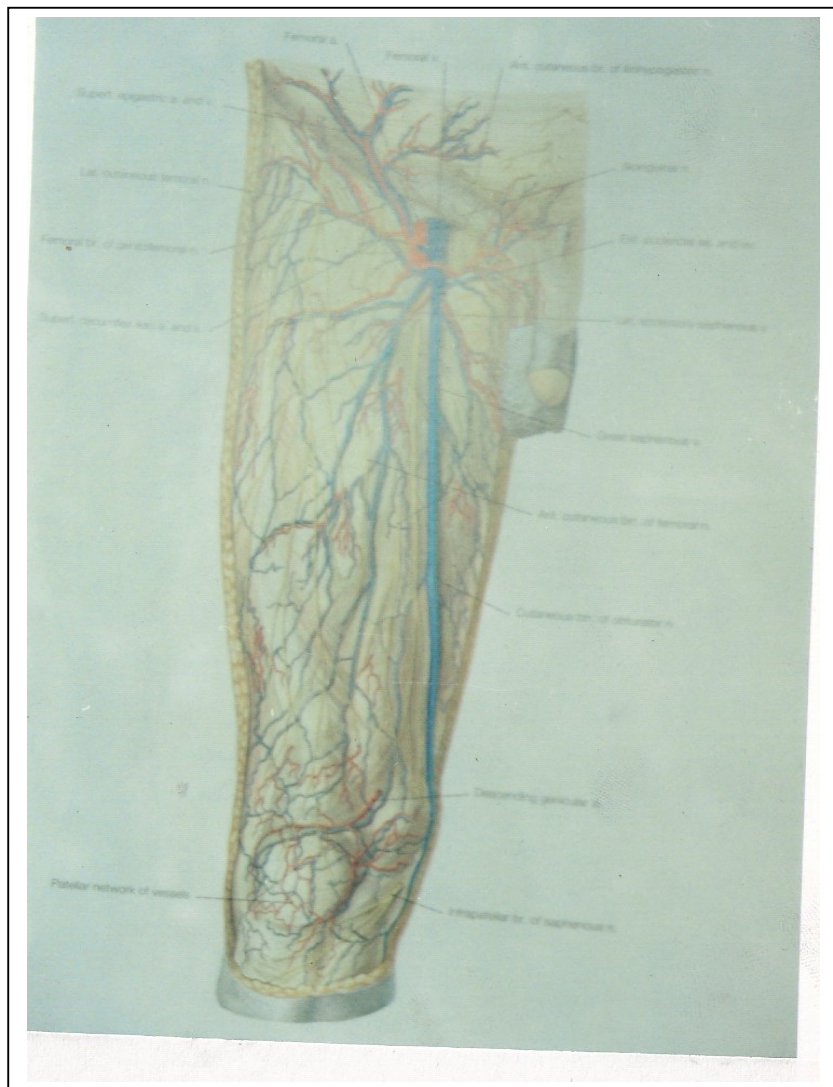
## CALLOUS OR CHRONIC ULCER



# ANATOMY OF VENOUS SYSTEM OF LOWER LIMB



## ANATOMY OF MAIN TRIBUTARIES OF GREAT SAPHENOUS VEIN



# ARTERIAL ULCER



## ANATOMY OF ARTERIES OF LOWER LIMB





## **TROPHIC ULCER**



## **ULCER IN CONGENITAL HEMOLYTIC ANAEMIA**



## **TROPICAL ULCER – SHOWING UNDERMINED EDGES**



## **DIABETIC ULCER**



## MADURAMYCOSIS

(Showing multiple discharging sinuses)



## SQUOMOUS CELL CARCINOMA OF LEG





## **FUNGATING SOFT TISSUE SARCOMA OF THIGH**



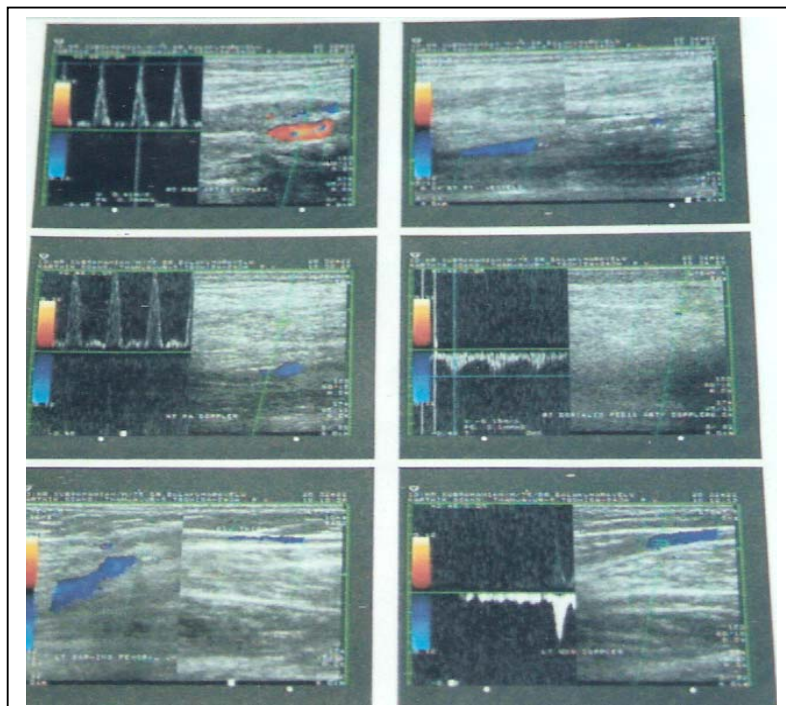
## **MALIGNANT MELANOMA OF FOOT**



MARJOLIN'S ULCER – A BURNT SCAR CONVERTING  
INTO SQUAMOUS CELL CARCINOMA



COLOR DOPPLER STUDY – SHOWING VARIOUS FLOW  
PATTERNS



## **VENOUS ULCER – SPLIT SKIN GRAFT DONE**

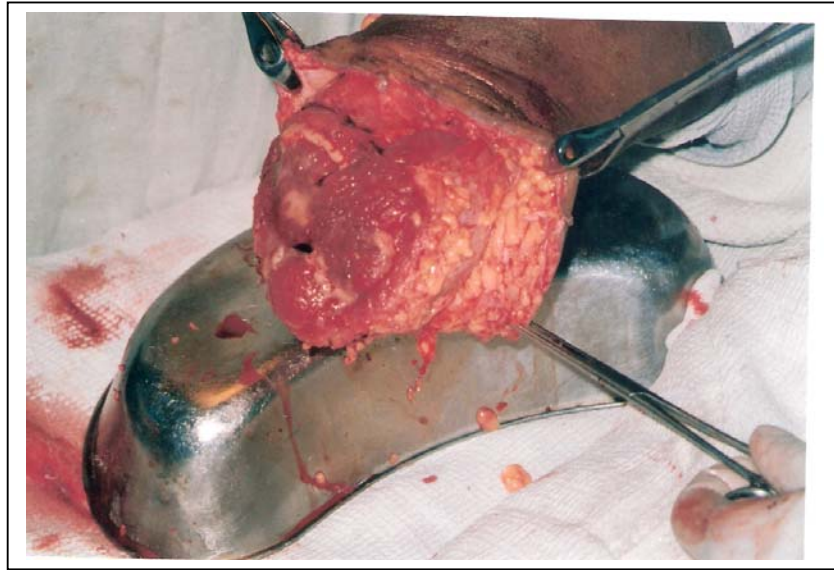


X RAY FOOT – showing osteomyelitic changes in great toe





## **BELOW KNEE AMPUTATION – Guillotine Amputation**



## **BK AMPUTATION – FLAP METHOD – Skin Incision**



## **BK AMPUTATION – FLAP METHOD**



## **BK AMPUTATION – SHOWING FLAP NECROSIS**











TYPE OF TREATMENT

